too, showed old pulmonary, glandular, or bone tuberculosis which had, in all probability, antedated the cirrhosis. The clinical picture is quite different from the alcoholic cases. The attacks of hepatic pain and the jaundice are absent. Indeed, the symptoms of any liver derangement are so slight as scarcely to draw attention to the organ. The importance of tuberculosis as an etiological factor in cirrhosis of the liver is further emphasized by certain animal experiments. Bensancon and Griffin, by the injection of pus from cold abscess, and Bartel by the injection of tubercle bacilli of low virulence, have observed in guinea-pigs, changes in the liver identical with cirrhosis as it occurs in man. Other authors have obtained similar results. Hansemann describes a form of spontaneous tuberculosis in guinea-pigs regularly associated with cirrhosis of the liver and often accompanied by attacks of jaundice.

Mechanism of Compensation in Tricuspid Insufficiency.—FRANKE (*Wien. klin. Woch.*, 1906, xix, 956). Many clinicians, considering the anatomical arrangement of the circulation, have contended that no satisfactory compensation of a tricuspid lesion can be established, as there is no driving force back of the lesion except the inefficient right auricle. As is shown clinically and experimentally, the left ventricle cannot overcome the deficiency as the right does in mitral lesions; in animals, after artificial damage to the tricuspid valves, the left ventricle is normal or a little atrophied. Franke cites cases to show that a certain degree of compensation does occur, that patients with serious tricuspid lesions may live comfortably for considerable periods of time, and that even after compensation has been broken, it may be satisfactorily re-established. He considers three factors as important in maintaining it. Hypertrophy of the right ventricle keeps up pressure in the pulmonary circulation and this pressure may be above normal, as is evidenced by the much accentuated second pulmonic sound. During diastole it exerts a suction action which assists the flow of blood from the auricle and veins. The elasticity and passive resistance of the auricle and large venous radicals prevent, to a certain degree, regurgitation. The contractile force of the hypertrophied right auricle, Franke thinks, is of small moment, as the presystolic wave formed in venous pulse tracings by its contraction is scarcely visible and certainly not accentuated above the normal. A very important feature, however, and a rather ingenious assumption is the position he gives the peripheral vessels in maintaining compensation. Hasebrock has drawn attention to what he terms the systole and the diastole of the peripheral vessels, and has pointed out its importance in sustaining the general circulation. Franke likens this action, which he thinks is accentuated by the passive congestion of tricuspid insufficiency, to the role the right ventricle plays in mitral lesions. This contractile power in the peripheral vessels, he believes, is most evident in the liver. The systolic regurgitant wave beating back into the veins finds its freest path along the hepatic vein, which is without valves. The greatest damming back, then, must occur in the liver, which is the meeting point of two venous waves coming in opposite directions; one the hepatic, the other the portal system. The pressure in the portal vein measures only from 7 to 13 mm. of mercury, while that in the hepatic, with an actively working hypertrophied right ventricle, must be decidedly higher. It is difficult to understand the absence of ascites in such cases unless there is some manner of regulation, and Franke ascribes such a self-regulation of the blood-flow to the liver itself. He assumes that, during the cardiac systole which is the diastole of the liver, blood is sucked in from the portal vein to be thrown with renewed force into the inferior vena cava by the succeeding contraction of the liver vessels and from here drawn into the right ventricle by its suction action.

The Intestinal Origin of Pulmonary Anthracosis.—SCHULTZE (*Münch. med. Woch.*, 1906, liii, 1702). The object of this article is to refute the conclusion of Calmette's students. This French school is accumulating more and more evidence in support of the intestinal origin of pulmonary tuberculosis, and recently van Steenberghe and Grysez have revived, from an experimental standpoint, Villoret's view of the intestinal origin of anthracosis. In their experiments on guinea-pigs and rabbits they mixed coal-dust and particles of India ink with the food, which, in some instances, they allowed the animals to eat, in others introduced directly into the stomach by means of a tube. At autopsy the pigment was found in the lungs and only in the lungs, the abdominal organs and mesenteric glands being free. The same result was obtained by intraperitoneal injections. In from twenty-four to forty-eight hours after an intraperitoneal injection of India ink they were able to demonstrate an anthracosis of the lungs and the tracheobronchial glands, while all other organs showed no pigmentation. Schultze, in repeating these experiments, comes to different results. After the intraperitoneal injections he finds a pigmentation of the lungs, but these are neither the only organs affected nor do they show any special predisposition toward its accumulation, for there is more pigment in the spleen and the liver than in the lungs. In his feeding experiments he obtained the same results as van Steenberghe and Grysez, the pigment being recovered only in the lungs, but his interpretation is different. He feels convinced that in feeding experiments, even when undertaken with the aid of a tube, it is impossible to exclude inhalation, and, indeed, this is how he explains its deposit in the lungs in these experiments. The fact that it is found only there, he believes, is almost convincing evidence as the distribution is so different when it is spread by the blood-stream. In a rabbit with a gastrostomy fistula he introduced pigments into the stomach over a period of two months and at section was able to find no deposit in the lungs.

Estimation of the Functional Capacity of the Heart.—FRITZ LEVY (*Ztschr. f. klin. Med.*, 1906, lx, 64). Ketzenstein has shown experimentally that when a large section of the arterial tree is suddenly cut off there is a rise of blood-pressure, and he has applied this principle to the functional study of the heart in man. The method is to determine the pulse-rate and the systolic blood-pressure in the brachial arteries and then compress by digital pressure the femoral arteries. Normally, a rise of blood-pressure of from 5 to 15 mm. of mercury occurs, while the pulse-rate remains constant or falls a little. Under certain conditions this rise is absent or there may even be a fall in the pressure associated with an increase in pulse-rate, a condition which Ketzenstein interprets as evidence of cardiac insufficiency. In cases of marked hypertrophy the